



PREDICTIVE VALUE OF BASELINE ARTERIAL LACTATE LEVELS FOR MORTALITY AND MORBIDITY IN PEDIATRIC HEAD TRAUMA

Medicine

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ABSTRACT

Introduction: Head traumas include the majority of trauma patients in pediatric emergency cases. The approach and follow-up to the traumatised patient are often complicated. In the present study, we aimed to evaluate the correlation between the predictive value of baseline arterial lactate levels and clinical indicators including Glasgow Coma Scale scores and positive computed tomography results and determine the effect of lactate on morbidity and mortality.

Material and Methods: In this prospective study, a hundred children ageing from 1 to 17 years, taken to the Pediatric Emergency Department for isolated head trauma without any chronic disorder were included between 1 January 2011 and 31 December 2014. Also, 40 healthy controls matched with age and gender were enrolled to compare the arterial lactate levels.

Results: Arterial lactate levels were considerably higher in the patient group compared to the controls. More importantly, lactate levels were significantly higher in cases with positive computed tomography results (haematoma, fracture) than cases with negative computed tomography results (3.944 ± 1.650 vs 1.578 ± 0.842). Similarly, lactate levels were significantly higher in hospitalized patients due to neurological sequel compared to non-hospitalized patients (4.385 ± 1.672 vs 1.750 ± 0.880). Also, there was a significant difference in lactate levels between the exitus group and survivors (5.608 ± 1.261 vs 2.359 ± 1.502). A significant positive correlation was found between the baseline lactate levels and Glasgow Coma Scale scores.

Conclusion: Based on the results obtained in the present study, we concluded that early lactate measurement has a significant predictive value for mortality and morbidity in pediatric head traumas.

KEYWORDS

Head trauma, Arterial lactate, Glasgow Coma Scale, Computed tomography.

INTRODUCTION

Head traumas include the majority of trauma patients in pediatric emergency cases. Traumatic brain injury (TBI) is a major cause of death and acquired disability among children in developed countries (1,2). Every year more than 50,000 individuals die due to traumatic brain injury, and additional 70,000-90,000 individuals are permanently disabled (3). World Health Organization predicts that by the year 2020 traffic deaths will be the third greatest cause of injury, and traumatic brain injury mainly occurs because of traffic accidents (4). Thus, while there is no cure for traumatic brain injuries, it is important to reduce the damage that can occur in the brain as a result of a traumatic injury (3).

Management of head injury begins in the pre-hospital setting and continues at the Emergency Department (ED) and intensive care unit (ICU) and aimed to maintenance of an adequate airway, oxygenation, ventilation and suitable arterial blood pressure to ensure an optimum cerebral perfusion pressure (5,6).

Previous studies demonstrated that lower initial Glasgow Coma Scale (GCS), to be <2 years old, presence of hypotension, hypoxia on admission, and subarachnoid haemorrhage, diffuse axonal injury and brain swelling on brain imaging are independent predictors of poor outcomes of head injury (7,8). However, the utility of metabolic alterations in prognostication of pediatric TBI is not well known.

In a previous study by Rahimi et al., it has been reported that initial mixed metabolic and respiratory acidosis is a significant predictor for mortality (9). Other investigators have demonstrated that initial base deficit and lactate levels were independent prognostic factors of mortality in severe pediatric trauma patients. However, there is not detailed information about the utility of these values in cases of pediatric TBI (10-13).

It has been reported that lactate is a metabolic waste product which indicates the potentially poor outcome. It is released during anaerobic metabolism and is, therefore, a marker of hypoperfusion and shock. In addition to ischemia and hypoxia, lactate may be elevated due to other etiologies, including circumstances that may either increase lactate production (such as white blood cells or certain medications) or decrease its clearance (such as liver or kidney failure) (14). Lactate's inability to correlate with other indicators of injury severity and to predict poor clinical outcome has also been reported (15).

Several studies showed that measurements of blood lactate levels

could be correlated to the outcome (16-18) or morbidity (19) after trauma. Elevated blood lactate levels are supposed to be secondary to anaerobic metabolism and thus it may reflect the degree of tissue hypoxia after injury.

Therefore, we aimed to design this prospective study to investigate the utility of arterial lactate in predicting mortality and morbidity and to evaluate its correlations with clinical indicators in patients with isolated head trauma

MATERIAL AND METHODS

In this prospective study, 100 children ageing from 1 to 17 years, taken to the Pediatric Emergency Department for isolated head trauma were included between 1 January 2011 and 31 December 2014. The study was approved by the Ethic Committee (102/2010). Children with any chronic disorder and diagnosed with any other disorder during the head trauma were excluded. Demographics, trauma mechanism, symptoms, radiological imaging results including direct head radiography and brain Computed Tomography (CT), Glasgow Coma Scale (GCS) scores, arterial lactate levels at admission and clinical outcomes including mortality and neurological disability were recorded. Forty healthy controls matched with age and gender were enrolled to compare the arterial lactate levels.

Data were reported as mean (SD), frequency and percentage. Mean differences and 95% confidence intervals (CIs) were used to compare the means. Pearson chi-square test and Fisher's exact test were used to analyze continuous and categorical data, respectively. Non-normally distributed data were compared by Mann Whitney U test. We used odds ratios (ORs) to determine the risk factors. The significance level was set at $P < 0.05$ and the advanced significance level was set at $P < 0.01$. All statistical tests were performed using SPSS V.11.0 software.

RESULTS

We enrolled 100 patients with isolated head trauma (39 girls, 39% and 61 boys, 61%) and 40 healthy controls (17 girls, 42.5% and 23 boys, 57.5%). The mean \pm SD age in the patients was 8.32 ± 4.90 years (range 0-18 years) and that in the controls was 7.15 ± 5.33 years. For patient group, 13 patients between 0-1 years, 36 were between 2-5 years, 33 were between 6-12 years, and 18 were between 13-18 years. Age and gender were not significantly different between the groups.

Trauma mechanisms were falls (53%), bicycle crash (13%), motor vehicle crash (11%), road car accident (6%) and the others (17%). We

assessed radiological imaging results of the head for all patients. Thirty-four patients had a fracture on direct head radiography. Forty-four patients had a positive (abnormal) brain CT scan, 56 had negative (normal) CT scan. Of positive CT scans, 43% of patients had a fracture, 34% had a haematoma, 23% had cerebral contusions. Sixteen patients had irritability, 33 had headache, 35 had nausea, 11 had seizure, and 33 had **loss of consciousness**. Sixty-four patients had scalp haematoma. Of them, 33 were >5 cm, 32 were <5 cm. Thirty-three patients were hospitalized after head trauma, and eight patients died. There were positive correlations among the hospitalization and trauma mechanism (falls), irritability, headache, nausea, scalp haematoma and positive CT scan results.

Mortality rate was significantly higher in patients with irritability (50%) than those without irritability (13%) ($p<0.01$). Also, it was significantly higher in patients with headache (87.5%) compared to those without headache (28.3%) ($p<0.01$). Similarly, the mortality rate was significantly higher in patients with loss of consciousness (87.5%) and positive CT scan results (100%) ($p<0.01$ for both). Odds ratios for mortality were higher in patients with irritability (OR=6.66), headache (OR=17.85), loss of consciousness (OR=17.85) and positive CT scan result (OR=1.31). Thirty-three patients had positive neurological examination finding, but the presence of positive neurological examination finding at admission was not associated with mortality ($p=0.828$).

The mean GCS score at admission was 13.40 ± 3.02 in patients with head trauma. The hospitalization rate well correlated with the GCS score, it was significantly higher in hospitalized patients (14.701 ± 0.628 vs 12.636 ± 2.826) ($p<0.001$). Similarly, it was found significantly lower in survivors than exitus patients (10.000 ± 3.703 vs 14.370 ± 1.238) ($p<0.01$). Also, patients with positive CT scan results had significantly lower GCS scores compared to patients with negative CT scan results (13.045 ± 2.579 vs 14.786 ± 0.530) ($p<0,01$).

The mean arterial lactate levels were 3.365 ± 2.284 in patients with head trauma and 1.356 ± 0.223 in the controls. There was a significant difference in arterial lactate levels between the patients and the controls. For patient group, lactate level was significantly higher in hospitalized patients due to a neurological sequel than non-hospitalized patients (4.385 ± 1.672 vs 1.750 ± 0.880) ($p=0.000$). It was considerably lower in survivors (2.359 ± 1.502 vs $5,608 \pm 1,261$) ($p=0.000$). Also, patients with positive CT scan results had significantly higher lactate values (3.944 ± 1.650 vs 1.578 ± 0.842) ($p<0.01$). The table indicates the effects of arterial lactate and GCS scores on hospitalization, CT scan results and mortality in the patient group.

Table. The effects of arterial lactate and GCS scores on hospitalization, CT scan results and mortality in the patient group.

		Lactate (mean±SD)	GCS (mean±SD)	p
Hospitalization	Hospitalized (n=33)	$4,385 \pm 1,672$	$12,636 \pm 2,826$	$0,000^{**}$
	Non-hospitalized (n=67)	$1,750 \pm 0,880$	$14,701 \pm 0,628$	$0,000^{**}$
CT scan result	Positive (abnormal) (n=44)	$3,944 \pm 1,650$	$13,045 \pm 2,579$	$0,000^{**}$
	Negative (normal) (n=56)	$1,578 \pm 0,842$	$14,786 \pm 0,530$	$0,000^{**}$
Mortality	Survivor (n=92)	$2,359 \pm 1,502$	$14,370 \pm 1,238$	$0,000^{**}$
	Exitus (n=8)	$5,608 \pm 1,261$	$10,000$ $3,703$	$0,000^{**}$

**Advanced significance ($p<0,01$)

DISCUSSION

In adults, head trauma and morbidity and mortality risks following severe traumatic brain injury (TBI) have been studied extensively, but such studies evaluated pediatric patients are limited (20-23) Early recognition of TBI at Emergency Department significantly affects the clinical outcome. Many researchers have been searching for newer diagnostic modalities to improve the management of head traumas. Measurement of arterial base deficit (BD) and lactate (LAC) has proven an easy and fast method for predicting morbidity, mortality and

resource consumption in patients with multiple trauma (24-26). Similarly, increased brain tissue or cerebrospinal fluid lactate (CSF-LAC) correlates with the severity of injury in patients with head trauma (27-30). Lannoo et al. (29) identified increased cerebral LAC as one of the predictors of mortality in patients with severe TBI. Goodman et al. (30) established an association between increased CSF-LAC levels (measured by microdialysis) and cerebral hypoxia and ischaemia. DeSalles et al. (28) found that patients with poor outcomes following head injury had significantly higher ventricular CSF-LAC levels than those with moderate disabilities or good outcomes. Wagner et al. (31) showed a high increase in LAC concentration in the white matter within the territory of vasogenic oedema of contused area shortly after experimental head injury in an animal model. Despite the availability of reasonable evidence on correlating cerebral LAC levels with severity of TBI, the diagnostic performance of arterial LAC and its ability in patients with isolated head injury has been inadequately studied.

Therefore, we performed this prospective study to determine the relationship between isolated head trauma, GCS and arterial lactate in children. Our results demonstrated that arterial lactate well correlated with mortality and morbidity in traumatized children. Arterial lactate levels were considerably higher in the patient group compared to the controls. More importantly, lactate levels were significantly higher in cases with positive computed tomography results (haematoma, fracture) than cases with negative computed tomography results. Similarly, lactate levels were significantly higher in hospitalized patients due to neurological sequel compared to non-hospitalized patients. Also, there was a significant difference in lactate levels between the exitus group and survivors. A significant positive correlation was found between the baseline lactate levels and Glasgow Coma Scale scores measured at admission.

It has been reported that blood lactate was a good predictor of mortality and morbidity. Increased blood lactate levels are secondary to the development of anaerobic metabolism, and reflect an imbalance between oxygen demand and oxygen supply. (32). Other factors may be involved in the elevation of lactate levels, including a reduced lactate elimination primarily by the liver, and the development of other cellular abnormalities altering pyruvate metabolism. (33). Nevertheless, in acute trauma, tissue hypoxia is likely to represent the primary mechanism responsible for elevated blood lactate levels (34). Arterial lactate measured at admission of emergency department was significantly higher in patients who died than in patients who survived. A significant correlation between high lactate levels and fatal outcome has been reported in critically ill patients with various types of pathologies 2527 including trauma. Siegel et al. (16) observed that the blood lactate level on admission was the most significant indicator of survival after blunt trauma. Milzam et al. (17) and Moyer et al. (35) also observed that the admission lactate level independently predicted mortality after trauma. Vincent et al (36) reported that blood lactate on the second day after admission was the only discriminator of survival. In a recent study on 76 patients, Abramson et al. (18) reported that initial lactate levels were not significantly higher in the nonsurvivors than the survivors, but the duration of hyperlactatemia was significantly longer in those patients.

Based on the literature and our results, we concluded that early lactate measurement has a significant predictive value for mortality and morbidity in pediatric head traumas. Arterial lactate measurement in children with isolated head trauma may be a simple strategy that may assist emergency medicine physicians to manage the traumatic brain injuries more promptly.

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Ethical approval

This study was approved by Eskisehir Osmangazi University Ethics Committee (No:2010/102) and performed according to the principles of Helsinki Declaration.

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